V. Adopting Healthful Lifestyle Habits to Lower LDL Cholesterol and Reduce CHD Risk

1. Population approach: promoting a base of healthy life habits

NCEP advocates a two-pronged approach for reducing CHD risk: the population approach and the clinical strategy. The two are closely linked. The population approach, which is outlined in the 1990 report of the Population Panel (National Cholesterol Education Program 1990; Report of the Expert Panel on Population Strategies . . . 1991), is designed to lower risk in the whole population through adoption of healthy life habits including a healthy diet, weight control, and increased physical activity. The clinical strategy is described in the ATP reports. This section summarizes the population approach and connects it to the clinical strategy. The clinical management team must recognize that they are an integral part of the population approach and contribute to it by providing education and guidance to the patient with high serum cholesterol and the patient's family.

The health community has provided the American public with consistent messages on cardiovascular risk reduction for the past four decades. These messages have encouraged avoidance or cessation of cigarette smoking, reduction of intakes of saturated fats and cholesterol, achieving and maintaining a healthy body weight, regular physical activity, and routine medical check-ups for blood pressure and cholesterol. Table V.1–1 (derived from the Healthy People 2010 publication) (U.S. Department of Health and Human Services 2000) reports the current status of the U.S. population on various healthy lifestyle habits and compares it with the goals for 2010.

Table V.1–1. Status Report on Healthy Lifestyle Habits: Healthy People 2010

Lifestyle Habit	Status in the 1990s	Goal for 2010
Healthy weight (BMI <25 kg/m^2)	42%	60%
Saturated fat intake <10% calories	36%	75%
Vegetable intake of at least 3 servings/day with at least 1/3 dark green or orange	3%	50%
Fruit intake of at least 2 servings/day	28%	75%
Grain intake of at least 6 servings/day with at least 1/3 whole grain	7%	50%
Smoking cessation by adult smokers	41%	75%
Regular physical activity of moderate intensity	15%	30%

Although progress has been made, it is clear that much more is needed to bring about the changes required to achieve the goals for 2010. The physician has an important role to play in this effort to help attain these goals.

The NHLBI, American Heart Association, and other organizations have mounted a major effort to reduce risk factors for CHD in the United States. Not only is there continuing research on improved methods for risk reduction, but national educational programs have also been put into effect. Table V.1–2 lists some of the Web sites of the programs sponsored by the U.S. Government.

Table V.1–2. Government-Sponsored Web Sites for Public Information: An Effective Way to Implement the Public Health Approach

Diet	www.nhlbi.nih.gov/chd www.nhlbi.nih.gov—then click on Aim for a Healthy Weight www.nhlbi.nih.gov/hbp www.nutrition.gov
Physical activity	www.surgeongeneral.gov/ophs/pcpfs.htm
Body weight	www.nhlbi.nih.gov—then click on Aim for a Healthy Weight
Cholesterol	www.nhlbi.nih.gov/chd
Blood pressure	www.nhlbi.nih.gov/hbp
Smoking cessation	www.cdc.gov/tobacco/sgr_tobacco_use.htm

Physicians and other health professionals have the opportunity to implement the public health and clinical approaches to risk reduction through interaction with patients and their families. Even in persons who are not candidates for clinical management of high serum cholesterol, control of other risk factors and preventive efforts convey the broader public health message to the patient. The physician's advice is valued and considered more credible than mass media or non-targeted educational campaigns. The physician can affect the public health arena in many ways. Table V.1–3 compares the role of the physician and other health professionals in the implementation of the public health approach with their role in the clinical management of risk factors through lifestyle changes.

Table V.1–3. The Role of the Physician and Other Health Care Professionals in Implementing the Population and Clinical Approaches to Lifestyle Modification

	Population Approach	Clinical Approach
Principles	Promote change in lifestyle habits by serving as a role model to patients Provide general advice and access to credible sources of information regarding healthy lifestyle habits	Promote targeted changes in individual lifestyle to produce significant reductions in an individual patient's risk Initiate outcome measurements that will be tracked during scheduled follow-up visits Physicians, dietitians, and other relevant health professionals should go beyond monitoring adherence to actively helping individuals overcome barriers and promote new behaviors
Diet	Briefly assess dietary intake of saturated fat and cholesterol Promote U.S. Dietary Guidelines (population diet) using pamphlets/handouts and Food Guide Pyramid – emphasize food portions Provide shopping and food preparation pamphlets/handouts highlighting low saturated fat foods including reduced fat dairy products, leaner meats, lower fat ground meat, and reduced fat baked goods Make full use of office personnel to promote public health message	Promote ATP III TLC diet using: • Individualized diet counseling that provides acceptable substitutions for favorite foods contributing to a patient's elevated LDL level – counseling often best performed by a registered dietitian • Reinforcement of dietary principles during follow-up visits at which LDL response to diet is assessed • Consideration of readiness to change and level of motivation
Physical activity	Promote regular physical activity by taking a physical activity history Provide pamphlets/advice regarding general principles of physical activity Recommend 30 minutes/day of regular moderate intensity activity	Follow Surgeon General recommendations for physical activity (U.S. Department of Health and Human Services. Physical activity and health. . . 1996b). Promote regular physical activity for individuals using: • Specific recommendations to increase physical activity based on a patient's cardiac status, age, and other factors • Specific advice regarding how physical activity could be integrated into the patient's lifestyle • Follow-up visits to monitor physical activity level, and follow-up counseling regarding barriers to daily physical activity

Table V.1–3. (continued)

	Population Approach	Clinical Approach
Body Weight	Ensure that weight, height, and waist circumference are measured at every visit Promote prevention of weight gain: • Provide access to tables identifying height/weight categories for BMI in waiting room or exam room • Provide literature relating BMI to health outcomes • Provide literature explaining use of Nutrition Fact labeling to identify calorie content and recommended portion sizes of foods	Follow Obesity Education Initiative (OEI) guidelines for weight management (National Institutes of Health 1998a,b) Promote prevention of weight gain: • Calculate BMI for every patient at every visit • Anticipate high-risk times for weight gain (perimenopausal years, times of significant life stress) and counsel patient on ways to prevent weight gain • Follow-up visits to discuss success of weight gain prevention strategies Discuss 10% weight loss goals for persons who are overweight: • Discuss lifestyle patterns that promote weight loss • Portion control • Daily physical activity • Follow-up visits to examine weight/BMI and discuss barriers to adherence
Cholesterol	Ensure that all adults age 20 and over have their blood cholesterol measured and their results explained in keeping with ATP III guidelines Ensure children and first degree relatives of adults in whom a genetic lipoprotein disorder is suspected have cholesterol screening performed	Follow ATP III guidelines for detection, evaluation, and treatment of persons with lipid disorders
Blood Pressure	Ensure that all adults have their blood pressure measured and their results explained in keeping with JNC VI guidelines	Follow JNC VI guidelines for the detection, evaluation, and treatment of persons with high blood pressure (JNC VI 1997; Joint National Committee . . . 1997)

Table V.1–3. (continued)

	Population Approach	Clinical Approach
Smoking cessation	Ensure that all persons are aware of the health hazards of cigarette smoking by using posters/handouts in the waiting room Query all persons regarding their smoking habits on every visit	Follow U.S. Department of Health and Human Services Clinical Practice Guideline: Treating Tobacco Use and Dependence (Public Health Service 2000) Promote smoking cessation: • Query regarding smoking habits • Provide targeted advice according to patient's knowledge base, e.g., dangers of smoking, benefits of quitting, and tips to quit • Schedule follow-up visits to discuss patient's progress in addressing smoking cessation

2. General approach to therapeutic lifestyle changes (TLC)

ATP III recommends a multifactorial lifestyle approach to reducing risk for CHD. This approach is designated *therapeutic lifestyle changes* (TLC) and includes the following components (see Table V.2–1):

- Reduced intakes of saturated fats and cholesterol
- Therapeutic dietary options for enhancing LDL lowering (plant stanols/sterols and increased viscous [soluble] fiber)
- Weight reduction
- Increased regular physical activity

Reduced intakes of saturated fats and cholesterol and other therapeutic dietary options for LDL-lowering (plant stanols/sterols and increased viscous fiber) are introduced first for the purpose of achieving the LDL cholesterol goal. After maximum reduction of LDL cholesterol is achieved with dietary therapy, emphasis shifts to management of the metabolic syndrome and its associated lipid risk factors (elevated triglycerides and low HDL cholesterol). A high proportion of patients with the metabolic syndrome are overweight/obese and sedentary; for them, weight reduction therapy and physical activity guidance is required to obtain further CHD risk reduction beyond that achieved by LDL lowering. At all stages of dietary therapy, physicians are encouraged to refer patients to registered dietitians or other qualified nutritionists for *medical nutrition therapy,* which is the term for the nutrition intervention and guidance provided by a nutrition professional.

Table V.2–1. Essential Components of Therapeutic Lifestyle Changes (TLC)

Component	Recommendation
LDL-raising nutrients	
Saturated fats*	Less than 7% of total calories
Dietary cholesterol	Less than 200 mg/day
Therapeutic options for LDL lowering	
Plant stanols/sterols	2 grams per day
Increased viscous (soluble) fiber	10–25 grams per day
Total calories (energy)	Adjust total caloric intake to maintain desirable body weight/prevent weight gain
Physical activity	Include enough moderate exercise to expend at least 200 Kcal per day

* *Trans* fatty acids are another LDL-raising fat that should be kept at a low intake

ATP III recommendations for ranges of other macronutrient intakes in the TLC Diet are given in Table V.2–2. Note that the recommendation for total fat ranges from 25 percent to 35 percent of total calories. To improve overall health, ATP III's lifestyle therapies generally contain the recommendations embodied in the Dietary Guidelines for Americans 2000 (U.S. Department of Agriculture . . . 2000).

Table V.2–2. Macronutrient Recommendations for the TLC Diet

Component	Recommendation
Polyunsaturated fat	Up to 10% of total calories
Monounsaturated fat	Up to 20% of total calories
Total fat	25–35% of total calories*
Carbohydrate†	50–60% of total calories*
Dietary fiber	20–30 grams per day
Protein	Approximately 15% of total calories

* ATP III allows an increase of total fat to 35 percent of total calories and a reduction in carbohydrate to 50 percent for persons with the metabolic syndrome. Any increase in fat intake should be in the form of either polyunsaturated or monounsaturated fat.

† Carbohydrate should derive predominantly from foods rich in complex carbohydrates including grains—especially whole grains—fruits, and vegetables.

The overall composition of the TLC Diet is consistent with the recommendations of the Dietary Guidelines for Americans 2000 (Table V.2–3). The dietary principles delineated in the Dietary Guidelines need not and should not be sacrificed for the purpose of LDL lowering. Furthermore,

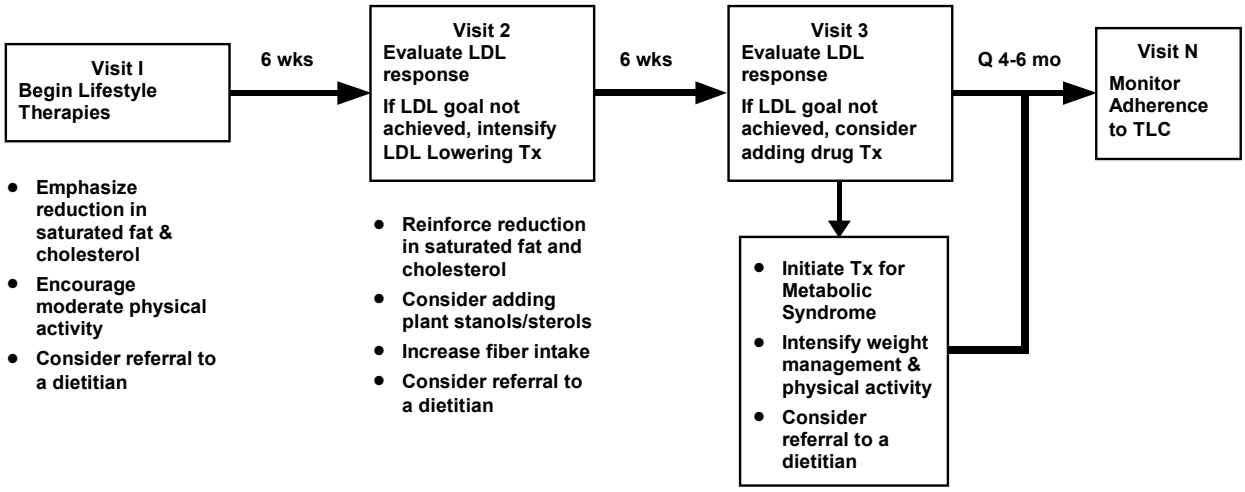
adherence to Dietary Guidelines recommendations should contribute to a reduction in risk beyond LDL lowering.

Table V.2–3. Dietary Guidelines for Americans 2000 (U.S. Department of Agriculture . . . 2000)

Aim for Fitness
• Aim for a healthy weight
• Be physically active each day
Build a Healthy Base
• Let the pyramid guide your food choices
• Choose a variety of grains daily, especially whole grains
• Choose a variety of fruits and vegetables daily
• Keep foods safe to eat
Choose sensibly
• Choose a diet that is low in saturated fat and cholesterol and moderate in total fat
• Choose beverages and foods to moderate your intake of sugars
• Choose and prepare foods with less salt
• If you drink alcoholic beverages, do so in moderation

Figure V.2–1 presents one model illustrating the general approach to dietary therapy.

Figure V.2–1. A Model of Steps in Therapeutic Lifestyle Changes (TLC)



During the first three months of dietary therapy, priority is given to lowering LDL cholesterol. In the first visit, the physician should address a few key questions and obtain an overall assessment of the individual's current life habits:

- Does the patient consume excess calories in the form of LDL-raising nutrients?
- Is the patient overweight or obese? Is abdominal obesity present?
- Is the patient physically active or inactive?
- If the patient is overweight/obese and/or physically inactive, is the metabolic syndrome present? (See Table II.6–1.)

To assess intakes of LDL-raising nutrients, the ATP III panel devised a brief Dietary CAGE that may be helpful (Table V.2–4). These questions are not a substitute for a systematic dietary assessment, which is usually carried out by a nutrition professional. CAGE questions can be used to identify the common food sources of LDL-raising nutrients—saturated fat and cholesterol—in the patient's diet. Also in the first visit, advice is given to begin moderate physical activity, but serious attempts to achieve weight loss can be delayed briefly to concentrate first on reducing intakes of LDL-raising nutrients. At any and every stage of dietary therapy, effective dietary modification will be facilitated by consultation with a registered dietitian or other qualified nutritionist for *medical nutrition therapy.* (Subsequently, the term ***nutrition professional*** will refer to a registered dietitian or qualified nutritionist.)

Table V.2–4. Dietary CAGE Questions for Assessment of Intakes of Saturated Fat and Cholesterol

- C—Cheese (and other sources of dairy fats—whole milk, 2% milk, ice cream, cream, whole fat yogurt)
- A—Animal fats (hamburger, ground meat, frankfurters, bologna, salami, sausage, fried foods, fatty cuts of meat)
- G—Got it away from home (high-fat meals either purchased and brought home or eaten in restaurants)
- E—Eat (extra) high-fat commercial products: candy, pastries, pies, doughnuts, cookies

After approximately 6 weeks, the physician should evaluate the LDL cholesterol response. If the LDL cholesterol goal has been achieved, or if progress in LDL lowering has occurred, dietary therapy should be continued. If the LDL goal is not achieved, the physician has several options to enhance LDL lowering. First, dietary instructions can be reexplained and reinforced. The assistance of a nutrition professional for more formal instruction and counseling (medical nutrition therapy) is especially valuable at this time. Second, therapeutic dietary options for LDL lowering (plant stanols/sterols and increased viscous fiber) will also enhance LDL lowering. Plant stanols/sterols are currently incorporated into special margarines, which are available directly to consumers. The stanol/sterol contents are listed on the food label. They may be available in other products in the future. Viscous fiber can be increased by emphasizing certain foods: cereal grains, fruits, vegetables, and dried beans, peas, and legumes (see Table V.2–5).

Table V.2–5. Food Sources of Viscous (Soluble) Fiber

Food Source	Soluble Fiber (g)	Total Fiber (g)
Cereal Grains (½ cup cooked)		
• Barley	1	4
• Oatmeal	1	2
• Oatbran	1	3
• Seeds		
– Psyllium Seeds, Ground (1 Tbsp)	5	6
Fruit (1 medium fruit)		
• Apples	1	4
• Bananas	1	3
• Blackberries (½ cup)	1	4
• Citrus Fruit (orange, grapefruit)	2	2-3
• Nectarines	1	2
• Peaches	1	2
• Pears	2	4
• Plums	1	1.5
• Prunes (¼ cup)	1.5	3
Legumes (½ cup cooked)		
• Beans		
– Black Beans	2	5.5
– Kidney Beans	3	6
– Lima Beans	3.5	6.5
– Navy Beans	2	6
– Northern Beans	1.5	5.5
– Pinto Beans	2	7
• Lentils (yellow, green, orange)	1	8
• Peas		
– Chick Peas	1	6
– Black Eyed Peas	1	5.5
Vegetables (½ cup cooked)		
• Broccoli	1	1.5
• Brussels Sprouts	3	4.5
• Carrots	1	2.5

After another 6 weeks, the response to dietary therapy should be evaluated. If the LDL cholesterol goal is achieved, the current intensity of dietary therapy should be maintained indefinitely. If the patient is approaching the LDL goal, consideration should be given to continuing dietary therapy before adding LDL-lowering drugs. If it appears unlikely that the LDL goal will be achieved with dietary therapy alone, drug therapy should be considered (see Section IV).

Thereafter, the metabolic syndrome, if present, becomes the target of therapy (see Section II). First-line therapy for the metabolic syndrome is weight control and increased physical activity. Again, referral to a nutrition professional for medical nutrition therapy to assist in weight reduction is recommended.

Finally, long-term monitoring for adherence to TLC is required. Revisits are indicated every 4–6 months during the first year of therapy and every 6–12 months in the long term. If a person is started on drug therapy, more frequent visits are advised.

The information shown in Table V.2–6 may be helpful for the physician both for dietary and lifestyle assessment and for guidance of the patient adopting TLC recommendations. The table is compiled from current ATP III dietary recommendations, Dietary Guidelines for Americans (U.S. Department of Agriculture . . . 2000), Obesity Education Initiative (OEI) guidelines for weight reduction (National Institutes of Health 1998a,b), and the Surgeon General's report on physical activity (U.S. Department of Health and Human Services 1996b).

Table V.2–6. Guide to Therapeutic Lifestyle Changes (TLC)

Healthy Lifestyle Recommendations for a Healthy Heart

Food Items to Choose More Often	Food Items to Choose Less Often	Recommendations for Weight Reduction	Recommendations for Increased Physical Activity
Breads and Cereals ≥6 servings per day, adjusted to caloric needs Breads, cereals, especially whole grain; pasta; rice; potatoes; dry beans and peas; low fat crackers and cookies **Vegetables** 3–5 servings per day fresh, frozen, or canned, without added fat, sauce, or salt **Fruits** 2–4 servings per day fresh, frozen, canned, dried **Dairy Products** 2–3 servings per day Fat-free, ½%, 1% milk, buttermilk, yogurt, cottage cheese; fat-free & low-fat cheese **Eggs** ≤2 egg yolks per week Egg whites or egg substitute **Meat, Poultry, Fish** ≤5 oz per day Lean cuts loin, leg, round; extra lean hamburger; cold cuts made with lean meat or soy protein; skinless poultry; fish **Fats and Oils** Amount adjusted to caloric level: Unsaturated oils; soft or liquid margarines and vegetable oil spreads, salad dressings, seeds, and nuts **TLC Diet Options** Stanol/sterol-containing margarines; viscous fiber food sources: barley, oats, psyllium, apples, bananas, berries, citrus fruits, nectarines, peaches, pears, plums, prunes, broccoli, brussels sprouts, carrots, dry beans, peas, soy products (tofu, miso)	**Breads and Cereals** Many bakery products, including doughnuts, biscuits, butter rolls, muffins, croissants, sweet rolls, Danish, cakes, pies, coffee cakes, cookies Many grain-based snacks, including chips, cheese puffs, snack mix, regular crackers, buttered popcorn **Vegetables** Vegetables fried or prepared with butter, cheese, or cream sauce **Fruits** Fruits fried or served with butter or cream **Dairy Products** Whole milk/2% milk, whole-milk yogurt, ice cream, cream, cheese **Eggs** Egg yolks, whole eggs **Meat, Poultry, Fish** Higher fat meat cuts: ribs, t-bone steak, regular hamburger, bacon, sausage; cold cuts: salami, bologna, hot dogs; organ meats: liver, brains, sweetbreads; poultry with skin; fried meat; fried poultry; fried fish **Fats and Oils** Butter, shortening, stick margarine, chocolate, coconut	**Weigh Regularly** Record weight, BMI, & waist circumference **Lose Weight Gradually** Goal: lose 10% of body weight in 6 months. Lose 1/2 to 1 lb per week **Develop Healthy Eating Patterns** • Choose healthy foods (see Column 1) • Reduce intake of foods in Column 2 • Limit number of eating occasions • Select sensible portion sizes • Avoid second helpings • Identify and reduce hidden fat by reading food labels to choose products lower in saturated fat and calories, and ask about ingredients in ready-to-eat foods prepared away from home • Identify and reduce sources of excess carbohydrates such as fat-free and regular crackers; cookies and other desserts; snacks; and sugar-containing beverages	**Make Physical Activity Part of Daily Routines** • Reduce sedentary time • Walk, wheel, or bike-ride more, drive less; Take the stairs instead of an elevator; Get off the bus a few stops early and walk the remaining distance; Mow the lawn with a push mower; Rake leaves; Garden; Push a stroller; Clean the house; Do exercises or pedal a stationary bike while watching television; Play actively with children; Take a brisk 10-minute walk or wheel before work, during your work break, and after dinner **Make Physical Activity Part of Exercise or Recreational Activities** • Walk, wheel, or jog; Bicycle or use an arm pedal bicycle; Swim or do water aerobics; Play basketball; Join a sports team; Play wheelchair sports; Golf (pull cart or carry clubs); Canoe; Cross-country ski; Dance; Take part in an exercise program at work, home, school, or gym

3. Components of the TLC diet

a. Major nutrient components

The major LDL-raising dietary constituents are saturated fat and cholesterol. A reduction in intakes of these components is the core of the TLC Diet. The scientific foundation for the relationship between high intakes of saturated fat and increased LDL levels dates back several decades and consists of several lines of evidence: observational studies, metabolic and controlled feeding studies, and clinical studies, including randomized clinical trials. These data have been reviewed in detail in previous reports of the NCEP (National Cholesterol Education Program 1993; 1994; National Cholesterol Education Program 1990; Report of the Expert Panel on Population Strategies . . . 1991), the U.S. Dietary Guidelines Committees (U.S. Department of Agriculture . . . 2000), and the American Heart Association (Krauss et al., 1996). The other major nutrients—unsaturated fats, protein, and carbohydrates—do not raise LDL cholesterol levels. In developing an LDL-lowering diet for ATP III, consideration was given not only to these long-established factors but also to new and emerging data that support the importance of the appropriate distribution of other nutrients that are related to cardiovascular health as well as general health. Therefore, the rationale for the recommendations for each component of the TLC diet will be described briefly.

1) Saturated fatty acids

Saturated fatty acids are a major dietary determinant of LDL cholesterol level (U.S. Department of Agriculture . . . 2000). The effects of saturated fatty acids on serum total cholesterol (and LDL cholesterol) levels have been studied extensively (Grundy and Denke, 1990). Several meta-analyses and reviews have been carried out to estimate the impact of saturated fatty acids on cholesterol levels (Mensink and Katan, 1992; Kris-Etherton and Yu, 1997). These analyses indicate that for every 1 percent increase in calories from saturated fatty acids as a percent of total energy, the serum LDL cholesterol rises about 2 percent. Conversely, a 1 percent reduction in saturated fatty acids will reduce serum cholesterol by about 2 percent. Recent trials confirm the efficacy of diets low in saturated fatty acids for lowering LDL levels. For example, the DELTA Study (Ginsberg et al., 1998) investigated the effects of reducing dietary saturated fatty acids from 15 percent of total calories to 6.1 percent of total calories. On the diet low in saturated fatty acids, LDL cholesterol was reduced by 11 percent. Another study, beFIT (Walden et al., 1997; 2000), tested effects of an NCEP therapeutic diet in individuals with hypercholesterolemia with and without hypertriglyceridemia. Compared to the participants' baseline diet, LDL cholesterol levels were reduced on the therapeutic diet by approximately 8 percent. Large-scale randomized controlled trials have been carried out to assess the safety of reduced intakes of saturated fatty acids and cholesterol in children and have found no evidence for compromised growth or development (Niinikoski et al., 1997; Obarzanek et al., 1997).

***Evidence statements**: There is a dose response relationship between saturated fatty acids and LDL cholesterol levels. Diets high in saturated fatty acids raise serum LDL cholesterol levels (A1). Reduction in intakes of saturated fatty acids lowers LDL cholesterol levels (A1, B1).*

The beneficial effects of reducing saturated fatty acids and cholesterol in the diet can be enhanced by weight reduction in overweight persons. Several studies have shown that LDL cholesterol levels can be lowered through weight reduction in overweight persons (National Institutes of Health 1998a,b). And most important, as shown in the MRFIT study, weight reduction will enhance serum cholesterol lowering brought about by a reduction in intakes of saturated fatty acids and cholesterol (Caggiula et al., 1981; Stamler et al., 1997).

Evidence statements: *Weight reduction of even a few pounds will reduce LDL levels regardless of the nutrient composition of the weight loss diet (A2), but weight reduction achieved through a calorie-controlled diet low in saturated fatty acids and cholesterol will enhance and sustain LDL cholesterol lowering (A2).*
Recommendation*: Weight loss through reduced caloric intake and increased levels of physical activity should be encouraged in all overweight persons. Prevention of weight gain also should be emphasized for all persons.*

Epidemiological studies show that populations that consume high amounts of saturated fatty acids and cholesterol have a high risk for CHD (Keys 1980, 1986). The evidence that lowering serum cholesterol levels by decreasing intakes of saturated fatty acids reduces the risk for CHD has been demonstrated in the meta-analysis by Gordon (1995a,b). This analysis included six robust dietary trials, in aggregate including 6,356 individuals. It showed that lowering serum cholesterol levels by reducing the intake of saturated fatty acids significantly decreased the incidence of CHD by 24 percent. There was also a trend toward a decrease in coronary mortality (21 percent) and total mortality (6 percent). No increase in non-CVD mortality was found.

The data from dietary trials, in combination with the results of controlled clinical trials with cholesterol-lowering medications (Gould et al., 1998; Brousseau and Schafer, 2000), document that reducing serum cholesterol and LDL cholesterol by diet alone or with pharmacological means will reduce CHD endpoints. The current American diet contains an average of about 11 percent of total calories as saturated fatty acids. The major sources of saturated fatty acids in the diet are high-fat dairy products (whole milk, cheese, butter, ice cream, and cream); high-fat meats; tropical oils such as palm oil, coconut oil, and palm kernel oil; and baked products and mixed dishes containing dairy fats, shortening, and tropical oils. To maximize LDL cholesterol lowering by reducing saturated fatty acid intake in the therapeutic diet, it will be necessary to lower intakes from the population mean intake of approximately 11 percent to <7 percent of total energy.

Evidence statements: *High intakes of saturated fatty acids are associated with high population rates of CHD (C2). Reduction in intake of saturated fatty acids will reduce risk for CHD (A1, B1).*
Recommendation: *The therapeutic diet to maximize LDL cholesterol lowering should contain less than 7 percent of total calories as saturated fatty acids.*

2) Trans fatty acid

Trans fatty acids are those in which double bonds are in the *trans* configuration. They are generally produced by hydrogenation of vegetable oils but some are found naturally in animal fats. Substantial evidence from randomized clinical trials indicates that *trans* fatty acids raise LDL cholesterol levels, compared with unsaturated fatty acids (Lichtenstein et al., 1993; 1999; Judd et al., 1994; 1998; Noakes and Clifton, 1998; Aro et al., 1997; Almendingen et al., 1995; Wood et al., 1993a,b; Nestel et al., 1992; Zock and Katan, 1992; Katan et al., 1995; Mensink and Katan, 1990). These studies also show that when *trans* fatty acids are substituted for saturated fatty acids, HDL cholesterol levels are lower (Ascherio et al., 1999), with a dose response effect observed. Recent United States data show that the use of liquid vegetable oil or semiliquid margarine results in the most favorable total and LDL cholesterol levels and ratios of total cholesterol to HDL cholesterol, whereas the use of butter or stick margarine results in the worst lipid levels (Lichtenstein et al., 1999). In addition, evidence from some epidemiological cohort studies suggests that high intakes of *trans* fatty acids are associated with higher risk for CHD (Kromhout 1995; Willett 1993; Pietinen 1997; and Hu 1999a). Whether this association is due to adverse effects of *trans* fatty acids on lipoproteins, to other adverse actions, or to confounding variables is uncertain.

The mean U.S. level of *trans* fatty acids intake is about 2.6 percent of total energy (compared with saturated fatty acids intake of ~11 percent of energy). Major sources of *trans* fatty acids in the diet include products made from partially hydrogenated oils such as baked products including crackers, cookies, doughnuts, breads, and products like french fries or chicken fried in hydrogenated shortening. Animal sources including dairy products provide smaller amounts of *trans* fatty acids. Soft margarines, tub and liquid, and vegetable oil spreads have low amounts of *trans* fatty acids. Some margarines and spreads are now *trans*-fatty acid free. Some hydrogenation of vegetable oils is the primary technology currently used to provide form to food products, so that they can be eaten out of the hand, rather than with a spoon.

Evidence statements: *Trans fatty acids raise serum LDL cholesterol levels (A2). Through this mechanism, higher intakes of trans fatty acids should increase risk for CHD. Prospective studies support an association between higher intakes of trans fatty acids and CHD incidence (C2). However, trans fatty acids are not classified as saturated fatty acids, nor are they included in the quantitative recommendations for saturated fatty acid intake of <7 percent of calories in TLC.*
Recommendation: *Intakes of trans fatty acids should be kept low. The use of liquid vegetable oil, soft margarine, and trans fatty acid-free margarine are encouraged instead of butter, stick margarine, and shortening.*

3) Dietary cholesterol

Dietary cholesterol causes marked hypercholesterolemia in many laboratory animals, including nonhuman primates. High intakes of cholesterol in humans, however, do not cause such a marked increase in serum cholesterol. Nonetheless, controlled metabolic studies in humans

indicate that high cholesterol intakes raise LDL cholesterol levels. The degree of rise varies from person to person, as is true for all nutrients. Meta-analyses of studies done in controlled settings confirm the LDL-raising action of dietary cholesterol (Hopkins 1992; Clarke et al., 1997). A recent meta-analysis showed that dietary cholesterol raises the ratio of total to HDL cholesterol, adversely affecting the serum cholesterol profile (Weggemans et al., 2001). A lesser effect of dietary cholesterol has been found in studies carried out in the outpatient setting (Howell et al., 1997); in this circumstance, failure to detect the full effect of dietary cholesterol is likely related to lack of tight metabolic control. On average, the response of serum cholesterol to dietary cholesterol as revealed in tightly controlled studies is approximately 10 mg/dL per 100 mg dietary cholesterol per 1000 Kcal (National Research Council 1989; Grundy et al., 1988).

In the past 40 years, there has been a progressive decline in intakes of dietary cholesterol. This has been the result of decreased intakes of eggs, high-fat meat, and high-fat dairy products. This reduction in cholesterol intake, along with a substantial reduction in the proportion of calories from saturated fatty acids, corresponds with the decline in serum cholesterol levels that has occurred in the U.S. population over four decades (Ernst et al., 1997). At present, the average U.S. daily consumption of cholesterol is 256 mg, higher for men (331mg) than for women (213 mg) (Tippett and Cleveland, 2000). Eggs contribute about one-third of the cholesterol in the food supply and this fraction has increased somewhat in recent years (Putnam and Gerrior, 1999). Other sources of dietary cholesterol include animal products, dairy, meats, poultry, and shellfish.

Evidence statements: *Higher intakes of dietary cholesterol raise serum LDL cholesterol levels in humans (A2, B1). Through this mechanism, higher intakes of dietary cholesterol should raise the risk for CHD. Reducing cholesterol intakes from high to low decreases serum LDL cholesterol in most persons (A2, B1).*
Recommendation: *Less than 200 mg per day of cholesterol should be consumed in the therapeutic diet to maximize the amount of LDL cholesterol lowering that can be achieved through reduction in dietary cholesterol.*

Some epidemiological data, namely the Western Electric Study, suggest dietary cholesterol increases heart disease risk independently of its effect on serum LDL cholesterol levels (Stamler and Shekelle, 1988). In contrast, data from two prospective cohort studies, the Nurses Health Study and the Health Professionals Study, found no significant association between frequency of reported egg consumption and CHD, except among diabetic women (Hu et al., 1999b).

4) Monounsaturated fatty acids

The most common form of monounsaturated fatty acids is oleic acid, which occurs in the cis form. Substitution of cis-monounsaturated fatty acids for saturated fatty acids results in a fall in LDL cholesterol levels (Mensink and Katan, 1992). Moreover, substitution of monounsaturated fatty acids for saturated fatty acids results in little or no decrease in HDL cholesterol and does not increase triglycerides as occurs with very high intakes of carbohydrates (>60 percent of total energy) (Mensink and Katan, 1992; Garg 1998,1992; Kris-Etherton 1999).

Monounsaturated fatty acids—as part of a diet that is low in saturated fatty acids and cholesterol and rich in vegetables, fruits, and grain products—have received increased attention as being potentially beneficial for risk reduction because of their association with low rates of CHD in olive-oil consuming populations of the Mediterranean basin (Keys et al., 1980, 1984, 1986). Despite epidemiological support for higher intakes of monounsaturated fatty acids, there are no controlled clinical trials that are designed to compare effects of monounsaturated and saturated fatty acids on CHD endpoints. This lack of data contrasts with several trials that replaced saturated fat with polyunsaturated fat.

Evidence statements: *Monounsaturated fatty acids lower LDL cholesterol relative to saturated fatty acids (A2, B2). Monounsaturated fatty acids do not lower HDL cholesterol nor raise triglycerides (A2, B2).*
Evidence Statement: *Dietary patterns that are rich in monounsaturated fatty acids provided by plant sources and rich in fruits, vegetables, and whole grains and low in saturated fatty acids are associated with decreased CHD risk (C1). However, the benefits of replacement of saturated fatty acids with monounsaturated fatty acids has not been adequately tested in controlled clinical trials.*
Recommendations: *Monounsaturated fatty acids are one form of unsaturated fatty acid that can replace saturated fatty acids. Intake of monounsaturated fatty acids can range up to 20 percent of total calories. Most monounsaturated fatty acids should be derived from vegetable sources, including plant oils and nuts.*

5) Polyunsaturated fatty acids

Polyunsaturated fatty acids, consisting mainly of n-6 linoleic acid, reduce LDL cholesterol levels when substituted for saturated fatty acids. At high intakes, linoleic acid also can produce small reductions in HDL cholesterol and triglycerides, although these responses are variable. Compared to cis-monounsaturated fatty acids, polyunsaturated fatty acids often cause a slightly greater reduction in LDL cholesterol levels (Mensink and Katan, 1992).

Several controlled clinical trials have compared the effects of polyunsaturated fatty acids, as a replacement for saturated fatty acids, on coronary endpoints (National Research Council 1989). Meta-analysis of trial results indicates that substitution of polyunsaturated fatty acids for saturated fatty acids reduces risk for CHD (Gordon 1995a,b; Mensink and Katan, 1992). This positive result is supported by research in primates that indicates that polyunsaturated fatty acids are antiatherogenic when substituted for saturated fatty acids (Rudel et al., 1995).

Despite evidence of CHD risk reduction from polyunsaturated fatty acids, there are no large populations that have consumed large quantities of polyunsaturated fatty acids for long periods. Thus, high intakes have not been proven safe in large populations; this introduces a note of caution for recommending high intakes.

Evidence statements: *Linoleic acid, a polyunsaturated fatty acid, reduces LDL cholesterol levels when substituted for saturated fatty acids in the diet (A1, B1). Polyunsaturated fatty acids can also cause small reductions in HDL cholesterol when compared with monounsaturated fatty acids (B2). Controlled clinical trials indicate that substitution of polyunsaturated fatty acids for saturated fatty acids reduces risk for CHD (A2, B2).*
Recommendations: *Polyunsaturated fatty acids are one form of unsaturated fatty acids that can replace saturated fat. Most polyunsaturated fatty acids should be derived from liquid vegetable oils, semi-liquid margarines, and other margarines low in trans fatty acids. Intakes of polyunsaturated fat can range up to 10 percent of total calories.*

6) Total fat

Among the fatty acids that make up the total fat in the diet, only saturated fatty acids and *trans* fatty acids raise LDL cholesterol levels (National Research Council 1989). Thus, serum levels of LDL cholesterol are independent of intakes of total fat per se. ATP II (National Cholesterol Education Program 1993, 1994) advised limiting total fat in Step I and Step II (TLC) diets to ≤30 percent of calories primarily as a means of achieving lower intakes of saturated fatty acids. The focus of the dietary approach to reducing CHD risk then and now is on dietary fatty acids that raise LDL cholesterol concentrations.

Evidence statement: *Unsaturated fatty acids do not raise LDL cholesterol concentrations when substituted for carbohydrates in the diet (A2, B2).*
Recommendation: *It is not necessary to restrict total fat intake for the express purpose of reducing LDL cholesterol levels, provided saturated fatty acids are reduced to goal levels.*

For many years, other public health groups have recommended low intakes of total fat in an effort to curtail obesity and to reduce the risk for some forms of cancer. These recommendations were based largely on experiments in laboratory animals and cross-cultural studies. Several short-term studies also suggest that higher fat intakes (>35 percent of calories) modify the body's metabolism in ways that favor fat accumulation (Astrup 1996; Astrup et al., 1997; Jeffery et al., 1995; Nelson and Tucker, 1996; Proserpi et al., 1997; Schutz 1995). However, isocaloric exchange of fat for carbohydrate does not produce weight gain over a period of many months (Hirsh et al., 1998; Leibel et al., 1992). Further, although some prospective studies have suggested a relationship between the percentage of dietary fat and obesity (Heitmann et al., 1995; Lissner and Heitmann, 1995), recent prospective studies (or meta-analysis of studies) have failed to detect a causative link between them (Seidell 1998; Willett 1998). Evidence related to these areas is reviewed in detail in the recent rationale report of the Dietary Guidelines for Americans (U.S. Department of Agriculture . . . 2000).

Studies in laboratory animals and cross-cultural studies have suggested a relationship between fat intake and risk for certain cancers (Greenwald et al., 1997; Harrison and Waterbor, 1999; Slattery et al., 1997a,b). Moreover, a major clinical trial is presently underway to determine

whether low-fat diets will reduce risk for breast cancer in women; this trial is a component of the Women's Health Initiative (Women's Health Initiative Study Group 1998) and is scheduled to end in 2005. Even so, recent prospective studies have not confirmed an association between fat intake and cancer (Giovannucci and Goldin, 1997; Holmes et al., 1999; Kolonel et al., 1999; Rose 1997). Thus, a strong recommendation to reduce fat intake for the purpose of preventing cancer does not seem warranted at this time (U.S. Department of Agriculture . . . 2000).

The Dietary Guidelines for Americans (U.S. Department of Agriculture . . . 2000) noted that some investigators are concerned that recommendations that emphasize lower total fat intakes (<30 percent energy) may have led to an overconsumption of carbohydrate, contributing to an increased prevalence of obesity. Moreover, very high intakes of carbohydrates (>60 percent of calories) in overweight/obese persons can aggravate some of the risk factors of the metabolic syndrome (Garg et al., 1998,1992; Chen 1995; Knopp et al., 1997; Grundy 1986; Mensink 1987). These latter responses have led some investigators to propose that populations with a high prevalence of insulin resistance and the metabolic syndrome should avoid very high-carbohydrate diets and should consume relatively more unsaturated fatty acids (Katan et al., 1997).

Evidence Statement: *The percentage of total fat in the diet, independent of caloric intake, has not been documented to be related to body weight or risk for cancer in the general population (U.S. Department of Agriculture . . . 2000). Short-term studies suggest that very high fat intakes (>35 percent of calories) modify metabolism in ways that could promote obesity (C2). On the other hand, very high carbohydrate intakes (>60 percent of calories) aggravate some of the lipid and nonlipid risk factors common in the metabolic syndrome (A2, B2, C2).*

Recommendations: *Dietary fat recommendations should emphasize reduction in saturated fatty acids. Further, for persons with lipid disorders or the metabolic syndrome, extremes of total fat intake—either high or low—should be avoided. In such persons, total fat intakes should range from 25–35 percent of calories. For some persons with the metabolic syndrome, a total fat intake of 30–35 percent may reduce lipid and nonlipid risk factors.*

7) Carbohydrate

When carbohydrates are substituted for saturated fatty acids, the fall in LDL cholesterol levels equals that with monounsaturated fatty acids. However, compared with monounsaturated fatty acids, substitution of carbohydrate for saturated fatty acids frequently causes a fall in HDL cholesterol and a rise in triglyceride (Mensink and Katan, 1992; Garg 1998; Turley et al., 1998; Knopp et al., 1997). This effect apparently persists in the long term, as suggested by differences in population lipid levels in the presence of different habitual diets (Knuiman et al., 1987; West et al., 1990). When carbohydrate is consumed along with high-fiber diets, however, the rise in triglycerides or fall in HDL cholesterol has been reported to be reduced (Jenkins et al., 1993; Turley et al., 1998; Vuksan et al., 2000).

Digestible carbohydrates include starches (complex carbohydrates) and sugar. Some foods, such as whole grains, vegetables, and some fruits, contain viscous fiber that helps to lower LDL cholesterol as well (see Table V.2–5). Sugars and starches occur naturally in many foods that

also supply other important nutrients. Examples of these foods include fat-free and low-fat dairy products, fruits, some vegetables, breads, cereals, and grains. Inclusion of these foods helps provide daily recommended intakes of essential nutrients (U.S. Department of Agriculture . . . 2000).

An old concept receiving recent attention is the "glycemic" potential of different foods. Glycemic index refers to the value obtained by feeding a carbohydrate load and measuring the level of blood glucose. Study of this factor is complicated because there is a wide range in the glycemic index for each group of foods, attributed to factors such as its form when eaten, the way it is processed, how it is chewed, how it is emptied from the stomach, and an individual's physiologic and metabolic responses (Levin 1999). To date the glycemic index has not been widely accepted as a practical means by which to select specific carbohydrate-containing foods for dietary therapy (U.S. Department of Agriculture . . . 2000).

Evidence Statement: *When carbohydrate is substituted for saturated fatty acids, LDL cholesterol levels fall (A2, B2). However, very high intakes of carbohydrate (>60 percent of total calories) are accompanied by a reduction in HDL cholesterol and a rise in triglyceride (B1, C1). These latter responses are sometimes reduced when carbohydate is consumed with viscous fiber (C2); however, it has not been demonstrated convincingly that viscous fiber can fully negate the triglyceride-raising or HDL-lowering actions of very high intakes of carbohydrates.*
Recommendation: *Carbohydrate intakes should be limited to 60 percent of total calories in persons with the metabolic syndrome. Lower intakes (e.g., 50 percent of calories) should be considered for persons with elevated triglycerides or low HDL cholesterol. Regardless of intakes, most of the carbohydrate intake should come from grain products, especially whole grains, vegetables, fruits, and fat-free and low-fat dairy products.*

8) Protein

Dietary protein in general has little effect on serum LDL cholesterol level or other lipoprotein fractions. However, substituting soy protein for animal protein has been reported to lower LDL cholesterol (Anderson 1995) (see Section V.3.b.3). Plant sources of protein are predominantly legumes, dry beans, nuts, and, to a lesser extent, grain products and vegetables, which are low in saturated fats and cholesterol. Animal sources of protein that are lower in saturated fat and cholesterol include fat-free and low-fat dairy products, egg whites, fish, skinless poultry, and lean meats.

b. Additional dietary options for LDL lowering

1) Increasing viscous fiber in the diet

Recent reports indicate that viscous (soluble) forms of dietary fiber can reduce LDL cholesterol levels. In contrast, insoluble fiber does not significantly affect LDL cholesterol (Anderson and Hannah, 1999). On average, an increase in viscous fiber of 5 to10 grams per day is accompanied by an approximately 5 percent reduction in LDL cholesterol (U.S. Department of Health and

Human Services 1997b; 1998a). In a meta-analysis of 67 trials related to oats, pectin, guar, and psyllium, a small but significant reduction in serum total and LDL cholesterol was noted for all sources of viscous fiber in ranges of 2–10 grams per day (Brown et al., 1999). Thus, at present, there is general agreement that viscous fiber (e.g., oats, guar, pectin, and psyllium) decreases serum cholesterol and LDL cholesterol. Because of the favorable effect of viscous fiber on LDL cholesterol levels, the ATP III panel recommends that the therapeutic diet be enriched by foods that provide a total of at least 5–10 grams of viscous fiber daily (see Table V.2–5). Even higher intakes of 10–25 grams per day can be beneficial.

Some investigators report that the consumption of viscous (soluble) fiber (provided by oats, barley, psyllium, pectin-rich fruit, and beans) produces a reduction in HDL cholesterol concentration (Anderson 1995). Other reviews report little, no, or inconsistent effect on HDL cholesterol (U.S. Department of Health and Human Services 1996; 1997a).

Evidence statement: *5–10 grams of viscous fiber per day reduces LDL cholesterol levels by approximately 5 percent (A2, B1).*
Recommendation: *The use of dietary sources of viscous fiber is a therapeutic option to enhance LDL cholesterol lowering.*

2) Plant stanols/sterols

Recent studies have demonstrated the LDL-lowering effect of plant sterols, which are isolated from soybean and tall pine-tree oils. Plant sterols can be esterified to unsaturated fatty acids (creating sterol esters) to increase lipid solubility. Hydrogenating sterols produces plant stanols and, with esterification, stanol esters. The efficacy of plant sterols and plant stanols is considered to be comparable (Hallikainen et al., 1999; Westrate and Meijer, 1998). Because lipids are needed to solubilize stanol/sterol esters, they are usually available in commercial margarines. The presence of plant stanols/sterols is listed on the food label. When margarine products are used, persons must be advised to adjust caloric intake to account for the calories contained in the products.

Data show that plant-derived stanol/sterol esters at dosages of 2 to 3 g/day lower LDL-C levels by 6 to 15 percent with little or no change in HDL cholesterol or triglyceride levels (Vuorio et al., 2000; Gylling and Miettinen 1999a; Gylling et al., 1997; Hallikainen et al., 1999; Hendriks et al., 1999; Miettinen et al., 1995; Vanhanen et al., 1993). The more recent among these studies indicate that maximal lowering of LDL cholesterol occurs at intakes of plant stanol/sterol esters of 2 g/day. LDL reductions also occur in individuals who have both hypercholesterolemia and type 2 diabetes (Gylling and Miettinen, 1994) and in children with hypercholesterolemia (Gylling et al., 1995). A greater percent lowering of LDL occurs in older people than in younger people (Law 2000). No studies have been conducted to determine the effect of plant stanols/sterols on CHD risk, although Law (2000) has recently projected that their use should double the beneficial effect on CHD risk achieved by reducing dietary saturated fatty acids and cholesterol.

Plant sterols/stanols reduce absorption of dietary carotenoids, and decreased levels of plasma beta-carotene have been observed subsequent to consumption of margarines that contain either stanol ester or sterol ester (Westrate and Meijer, 1998). Whether carotenoid decreases are deleterious is unknown, but prudence calls for adhering to current recommendations for intakes of fruits and vegetables with consumption of plant stanols/sterols.

Evidence Statement: *Daily intakes of 2–3 grams per day of plant stanol/sterol esters will reduce LDL cholesterol by 6–15 percent (A2, B1).*
Recommendation: *Plant stanol/sterol esters (2 g/day) are a therapeutic option to enhance LDL cholesterol lowering.*

3) Soy protein

Soy protein included in a diet low in saturated fatty acids and cholesterol can lower levels of total cholesterol and LDL cholesterol in individuals with hypercholesterolemia. Recent reviews (U.S. Department of Health and Human Services 1998b; 1999a) gave particular weight to 16 well-controlled trials that reported intakes of saturated fatty acids and cholesterol. More than half of the studies used more than 40 g soy protein/day in some form. One report (Jenkins 2000b) indicated that 25 g/day soy protein in a diet low in saturated fatty acids and cholesterol lowers LDL cholesterol levels by about 5 percent.

The specific processing of the soybean determines the characteristics of soy protein, such as the content of isoflavones, fiber, and saponins. There is some evidence that an LDL-lowering effect is dependent upon isoflavone content (Crouse et al., 1999) but conclusive data are lacking. Since there are inconsistent findings regarding both the dose and the potential benefit of soy protein, soy protein's major role in LDL-lowering may be to help reduce the intake of animal food products with their higher content of saturated fatty acids.

Evidence statement: *High intakes of soy protein can cause small reductions in LDL cholesterol levels, especially when it replaces animal food products (A2, B2).*
Recommendation: *Food sources containing soy protein are acceptable as replacements for animal food products containing animal fats.*

c. Other dietary factors that may reduce baseline risk for CHD

Epidemiological studies strongly suggest that other nutrient factors affect baseline risk for CHD. For example, in the Mediterranean region, where the diet is rich in fruits and vegetables, whole grains, ocean fish, and unsaturated fatty acids, the risk for CHD appears to be lower than predicted by the major risk factors. In contrast, in regions without this dietary pattern, such as Eastern Europe and Russia, CHD rates are higher than predicted by the prevalence of CHD risk factors. Such observational data provide a basis for a general recommendation for a dietary

pattern that is consistent with a low baseline population risk. The Dietary Guidelines for Americans, 2000, were crafted to facilitate reduction in baseline risk for CHD (Table V.2–3).

In addition, nutritional research has focused on several specific factors that may have unique properties to reduce risk for CHD. The status of these emerging dietary factors are reviewed below and summarized in evidence statements.

1) n-3 (omega-3) polyunsaturated fatty acids

Polyunsaturated fatty acids of the n-3 (omega-3) type occur as alpha-linolenic acid (18:3), primarily in certain vegetable sources such as soybean, canola oil and English walnuts, and in fish oils as eicosapentaenoic acid (EPA) (20:5) and docosahexaenoic acid (DHA) (22:6) (*marine n-3 fatty acids)*.

Moderate fish consumption has been associated with reduced sudden cardiac death or reduced CHD mortality in several prospective cohort studies (Albert et al., 1998; Daviglus et al., 1997; Dolecek and Grandits, 1991) but not in others (Morris et al., 1995; Ascherio et al., 1995). One study found a trend toward increased relative risk of CHD death with marine n-3 fatty acids. A nested, case-control study found an inverse relationship between risk for sudden cardiac death and both reported intake of marine n-3 fatty acids and red blood cell n-3 fatty acid level (Siscovick et al., 1995). Postulated mechanisms for the effects of marine n-3 fatty acids on CHD risk include favorable effects on cardiac rhythm, platelet aggregation, inflammatory responses, and serum triglyceride levels. High intakes of marine n-3 fatty acids reduce triglyceride levels (Roche 2000); this effect appears to be secondary to decreased VLDL production (Harris 1989). Generally, marine n-3 fatty acids have no effect on LDL cholesterol levels, but large doses have been shown to reciprocally increase LDL cholesterol levels in persons with hypertriglyceridemia (Harris 1997). Recent data indicate that some fish have a high mercury content and the toxic effects of mercury could attenuate protective effects of fish (National Research Council 1999; Rissanen et al., 2000).

Four clinical trials suggest that n-3 fatty acids from marine or plant sources reduce sudden death and overall death in populations with pre-existing cardiovascular disease. The DART trial (Burr et al., 1989) was a relatively large secondary prevention trial in which subjects advised to eat fatty fish had a 29 percent reduction in 2-year all-cause mortality compared with those not so advised, although myocardial infarction and coronary death were not specifically reduced. The Lyon Heart Trial (de Lorgeril et al, 1999) included increased intakes of alpha-linolenic acid as part of a "Mediterranean" diet. Compared to the control group, subjects consuming the Mediterranean diet had fewer coronary events. The authors attributed some of the benefit to higher intakes of n-3 fatty acids. In a small supplement trial, Singh et al. (1997) treated patients with suspected acute myocardial infarction with fish oil capsules (EPA 1.08 g/day) or mustard oil (alpha-linolenic acid 2.9 g/day) or placebo. After one year, total cardiac events were significantly less in the groups on fish oil and mustard seed oil supplements. Further, the large placebo-controlled, but unblinded Italian GISSI Prevention trial (GISSI-Prevenzione Investigators 1999) administered fish oil supplements containing n-3 fatty acids (1 g fish oil daily, n = 2836 subjects) and compared coronary outcomes to controls (n = 2828). The group receiving fish-oil supplements had a 14 percent reduction in total death and a 17 percent

reduction in cardiovascular death. Other clinical trials are less suggestive of benefit from n-3 fatty acids. Angiographic data fail to show that marine n-3 fatty acids modify coronary lumen size (von Schacky et al., 1999; Sacks 1995). Also, fish oil administration apparently does not prevent restenosis after coronary angioplasty (Leaf 1994). Additional studies are underway to determine the effect of n-3 fatty acids on CHD risk in the U.S. population (U.S. Department of Agriculture . . . 2000).

Based on these findings, the Dietary Guidelines for Americans (U.S. Department of Agriculture . . . 2000) noted that some fish, such as salmon, tuna, and mackerel, contain omega-3 fatty acids that are being studied to determine if they offer protection against heart disease. No quantitative recommendations for n-3 fatty acids were made for the general public.

Evidence statement: *The mechanisms whereby n-3 fatty acids might reduce coronary events are unknown and may be multiple. Prospective data and clinical trial evidence in secondary CHD prevention suggest that higher intakes of n-3 fatty acids reduce risk for coronary events or coronary mortality (A2, C2).*
Recommendation: *Higher dietary intakes of n-3 fatty acids in the form of fatty fish or vegetable oils are an option for reducing risk for CHD. This recommendation is optional because the strength of the evidence is only moderate at present. ATP III supports the American Heart Association's recommendation that fish be included as part of a CHD risk-reduction diet. Fish in general is low in saturated fat and may contain some cardioprotective n-3 fatty acids. However, a dietary recommendation for a specific amount of n-3 fatty acids is not being made (See Section VI for ATP III recommendations on n-3 supplements for reducing risk for CHD.)*

2) Vitamins/antioxidants

a) Folic acid and vitamins B_6 and B_{12}

Folic acid and vitamins B_6 and B_{12} play a role in the metabolism of homocysteine, and levels of these vitamins correlate inversely with homocysteine levels. Data from the Framingham Heart Study suggest that the mandated fortification of cereal grains with folic acid has lowered population mean homocysteine levels as well as the prevalence of hyperhomocysteinemia (Jacques et al., 1999). Many cross-sectional case-control studies and some prospective cohort studies show a positive association between plasma homocysteine levels and CVD risk (Nygard et al., 1997; Graham et al., 1997; Boushey et al., 1995; Perry et al., 1995; Arnesen et al., 1994; Stampfer et al., 1992) but other prospective cohort studies do not (Alfthan et al., 1994;Verhoef et al., 1997; Evans et al., 1997; Folsom et al., 1998).

Despite the fact that homocysteine levels can be reduced with supplements of folate, B_6, and B_{12}, it is not known whether reduction of plasma homocysteine levels by diet and/or vitamin supplements will reduce CVD risk (Stampfer and Malinow, 1992). Several randomized trials are underway to determine if folic acid, vitamin B_6, and vitamin B_{12} will be effective in reducing the risk of heart disease (Clark and Collins, 1998).

The Institute of Medicine has recently published dietary recommendations for folate for the general population (National Research Council 2000a). The recommended dietary allowance (RDA) for folate is 400 micrograms per day. This level of intake was deemed adequate to provide any reduction in risk for cardiovascular disease that can be obtained from dietary folate. An upper limit for folate derived from fortified food or supplements was estimated to be 1000 micrograms per day.

***Evidence statement**: According to the Institute of Medicine, the RDA for folate for adults is 400 micrograms per day, and the upper limit is 1000 micrograms per day. There are no published randomized controlled clinical trials to show whether lowering homocysteine levels through dietary intake or supplements of folate and other B vitamins will reduce the risk for CHD.*
***Recommendation:** ATP III endorses the Institute of Medicine RDA for dietary folate, namely, 400 micrograms per day. Folate should be consumed largely from dietary sources.*

b) Antioxidants

Oxidative stress is a putative cause of atherosclerotic disease. In experimental studies, oxidation of LDL is an important step in the development and progression of CHD. Thus, a large body of research has been directed towards the potential of antioxidants for reducing CHD risk. Antioxidants under investigation include ascorbic acid (vitamin C), alpha-tocopherol (vitamin E), beta-carotene, ubiquinone (coenzyme Q10), bioflavonoids, and selenium.

Several studies in laboratory animals support the concept that antioxidants are antiatherogenic (National Research Council 2000b). Some, but not all, epidemiological data lend additional support to the concept that dietary antioxidants can reduce risk for CHD (National Research Council 2000b). Generally, in populations that consume a dietary pattern rich in fruits and vegetables and other foods high in antioxidants, there is a reduced risk of CHD.

Several controlled clinical trials have been carried out to determine whether supplementation with antioxidants reduces risk for CHD. The Linxian study in China found that supplements of beta-carotene (15 mg/d), vitamin E (30 mg/d), and selenium (15 mcg/d), given at levels obtained from foods, were associated with a non-significant 10 percent decrease in CVD mortality (Blot et al., 1993). In the Alpha-Tocopherol, Beta Carotene Cancer Prevention Study, supplementation with beta-carotene had no beneficial effect on the incidence of myocardial infarction (Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group 1994). Another trial (Hennekens et al., 1996), found no benefit (or harm) for CHD incidence after 12 years of beta-carotene supplementation in 22,071 male physicians. Finally, in the CARET study, a non-significant 26 percent increase in cardiovascular mortality was reported in a group supplemented with beta-carotene (Omenn et al., 1996).

In the Alpha-Tocopherol, Beta Carotene Cancer Prevention Study, supplementation with small doses of vitamin E in Finnish male smokers had only a marginal effect on incidence of fatal CHD, whereas it had no effect on incidence of nonfatal myocardial infarction (Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group 1994). In a secondary prevention

trial among patients with CHD, vitamin E supplementation (400 or 800 IU per day during 1.5 years) in the Cambridge Heart Antioxidant Study (CHAOS), significantly reduced the risk for recurrent MI (77 percent). No effect was demonstrated for CVD mortality. A non-significant increase in total mortality was observed in the vitamin E group (Stephens, et al., 1996). Two large-scale clinical trials in patients with established CHD failed to demonstrate a protective effect of vitamin E supplementation on subsequent cardiovascular events (GISSI-Prevenzione Investigators 1999; Heart Outcomes Prevention . . . Investigators 2000a,b).

Thus, in spite of the theoretical benefits of antioxidant vitamins for reducing risk for CHD, this potential has so far not been found in controlled clinical trials that have used a variety of antioxidant mixtures and doses. The failure to demonstrate benefit in controlled trials does not eliminate the possibility of benefit. It does, however, dilute confidence in benefit and stands in the way of a solid recommendation for high intakes of antioxidants for CHD prevention.

The Institute of Medicine has recently released recommendations for Dietary Reference Intakes (DRIs) for antioxidant vitamins. A specific recommendation was not made for beta-carotene because it has not been shown to be an essential nutrient nor have clinical trials demonstrated benefit for reduction in risk for either cardiovascular disease or cancer. The RDA for vitamin C was increased to 75 mg/day for women and 90 mg/day for men. The RDA for Vitamin E was set at 15 mg/day. Vitamin E supplementation was not recommended for prevention of chronic disease because of a lack of convincing evidence of benefit.

Evidence Statement: *Oxidative stress and LDL oxidation appear to be involved in atherogenesis. However, clinical trials to date have failed to demonstrate that supplementation of the diet with antioxidants will reduce risk for CHD (A2).*

Recommendation: *Evidence of CHD risk reduction from dietary antioxidants is not strong enough to justify a recommendation for antioxidant supplementation to reduce CHD risk in clinical practice. ATP III supports current recommendations of the Institute of Medicine's RDAs for dietary antioxidants, i.e., 75 mg and 90 mg per day for women and men, respectively, for vitamin C and 15 mg per day for vitamin E.*

3) Moderate intakes of alcohol

Observational studies consistently show a J-shaped relation between alcohol consumption and total mortality. Moderate alcohol consumption is associated with lower mortality, and higher consumption with higher mortality. The lower mortality appears to be related to CHD death, because CHD accounts for a significant proportion of total deaths. Case-control, cohort, and ecological studies indicate lower risk for CHD at low to moderate alcohol intake (Criqui 1996). A moderate amount of alcohol can be defined as no more than one drink per day for women and no more than two drinks per day for men (Criqui 1987; Dufour 2001). This gender distinction takes into account differences in both weight and metabolism. Moreover, any cardiovascular benefit occurs not in the young age groups but in middle-aged adults, men 45 years of age or older and women 55 years of age or older (Thun et al., 1997). Mechanisms of putative risk reduction from moderate alcohol consumption are unknown; however, it could be due to an

increase in HDL cholesterol and apo A1 and modestly to an improvement in hemostatic factors (Rimm et al., 1999). Prospective cohort studies suggest a similar relationship with CHD regardless of the type of alcoholic beverages consumed (Rimm et al., 1996).

The dangers of overconsumption of alcohol are well known. At higher levels of intake, adverse effects include elevated blood pressure, arrhythmia, and myocardial dysfunction (Criqui 1987, 1996). Alcohol excess also predisposes to acute pancreatitis. Rarely it can precipitate pancreatitis by accentuating a pre-existing hypertriglyceridemia and chylomicronemia (Fortson et al., 1995). A pooled analysis shows that alcohol intake increases the risk of breast cancer in women (Smith-Warner et al., 1998). Since up to 10 percent of U.S. adults misuse alcohol, advice about alcohol intake should be given carefully with both advantages and negatives presented (Criqui 1998). For some persons, the negatives of alcohol consumption will outweigh any advantage.

Evidence Statement: *Moderate intakes of alcohol in middle-aged and older adults may reduce risk for CHD (C2). However, high intakes of alcohol produce multiple adverse effects (C1).*

Recommendation: *No more than two drinks per day for men and no more than one drink per day for women should be consumed. A drink is defined as 5 ounces of wine, 12 ounces of beer, or 1½ ounces of 80 proof whiskey. Persons who do not drink should not be encouraged to initiate regular alcohol consumption.*

4) Dietary sodium, potassium, and calcium

Many individuals with hypercholesterolemia also have hypertension (see Section VII.6). Evidence suggests that even those with normal blood pressure levels can reduce their chances of developing high blood pressure by consuming less salt (National Research Council 1989; JNC VI 1997; Joint National Committee . . . 1997). Studies in diverse populations have shown that a high sodium intake is associated with higher blood pressure (Chobanian and Hill, 2000). Also, a high salt intake increases the amount of calcium excreted in the urine, and has been independently associated with bone loss at the hip (Chobanian and Hill, 2000). The Dietary Approaches to Stopping Hypertension (DASH) trial has provided evidence that a dietary pattern high in fruits, vegetables, low-fat dairy products, whole grains, poultry, fish, and nuts and low in fats, red meat, and sweets—foods that are good sources of potassium, calcium, and magnesium–favorably influences blood pressure even when sodium levels are held constant (Appel et al., 1997), but when these nutrients are consumed in combination with a low sodium intake, 2400 mg or 1800 mg, blood pressure is lowered even more (Sacks et al., 2001).

Evidence Statement: *JNC VI (JNC VI 1997; Joint National Committee . . . 1997) provides a review of the evidence to support the concept that lower salt intake lowers blood pressure or prevents its rise. One clinical trial further shows that the effects of a dietary pattern high in fruits, vegetables, low-fat dairy products, whole grains, poultry, fish, and nuts and low in fats, red meat, and sweets—foods that are good sources of potassium, calcium, and magnesium—to reduce blood pressure are enhanced by a diet low in salt (A2).*

Recommendation: *The Diet and Health report (National Research Council 1989) and JNC VI recommend a sodium intake of <2400 mg/d (no more than 100 mmol/day, 2.4 g sodium or 6.4 g sodium chloride). JNC VI further recommends maintaining adequate intakes of dietary potassium (approximately 90 mmol per day) and enough dietary calcium and magnesium for general health. ATP III affirms these recommendations for persons undergoing cholesterol management in clinical practice.*

5) Herbal or botanical dietary supplements

The 10 top-selling herbal or botanical dietary supplements are cranberry, echinacea, evening primrose, garlic, ginkgo, ginseng, goldenseal, grape seed extract, St. John's wort, and saw palmetto (Borchers et al., 2000). These botanical supplements are available in health food stores, pharmacies, and many supermarkets. Several of the compounds have been promoted as agents to reduce the risk of CHD. Data from controlled trials regarding efficacy and safety are limited, in part because existing food and drug laws do not require demonstration of safety and efficacy to support legal marketing of dietary supplements. Dietary supplements are regulated according to different standards than are drugs. In addition to concerns about efficacy and safety, there is a lack of standardization among brands of botanical supplements. As a result, the amount of bioactive constituent, by which the supplements are hypothesized to influence disease, can differ widely among brands. In the case of garlic, a few randomized controlled studies are available, but the preponderance of available evidence fails to establish that garlic reduces LDL cholesterol levels. Biological plausibility supports use of some supplements, but there are few controlled clinical trials to document benefit. Studies designed to evaluate efficacy for disease endpoints, long-term safety, and drug interaction have not been reported.

Evidence statement: *Despite widespread promotion of several herbal or botanical dietary supplements for prevention of CHD, a paucity of data exists on product standardization, controlled clinical trials for efficacy, and long-term safety and drug interactions. Clinical trial data are not available to support the use of herbal and botanical supplements in the prevention or treatment of heart disease.*

Recommendation: *ATP III does not recommend use of herbal or botanical dietary supplements to reduce risk for CHD. However, health care professionals should query patients to establish whether such products are being used because of the potential for drug interaction.*

6) High protein, high total fat and saturated fat weight loss regimens

Periodically, weight-loss diets high in protein and fat and low in carbohydrate surge in popularity. Such diets will result in weight loss within a few weeks or months if calories are restricted. However, such diets have not been demonstrated to produce long-term weight loss in controlled trials. Although clinical trial data are lacking, several concerns have been expressed about the use of these diets in clinical weight reduction:

- Short-term, extreme diets rarely produce long-term weight reduction.

- High intakes of saturated fats can raise LDL cholesterol.
- Low intakes of fruits, vegetables, and grains can deprive persons of healthful nutrients and are not conducive to long-term health.

Diets popularized as low-carbohydrate, high-fat, high-protein regimens for rapid weight loss should not be confused with ATP III's easing restriction of the percentage of dietary fat for persons with the metabolic syndrome. The latter allows dietary fat to rise to 35 percent of total calories, provided it remains low in saturated fatty acids (<7 percent of total energy) and includes mostly unsaturated fats. This will reduce carbohydrate intake somewhat to prevent the actions of high-carbohydrate diets to raise triglycerides and reduce HDL cholesterol levels. The ATP III recommendation allows for the dietary variety outlined in Dietary Guidelines for Americans 2000 (U.S. Department of Agriculture . . . 2000).

Evidence statement: *High protein, high total fat and saturated fat weight loss regimens have not been demonstrated in controlled clinical trials to produce long-term weight reduction. In addition, their nutrient composition does not appear to be conducive to long-term health.*
Recommendation: *High protein, high total fat and saturated fat weight loss regimens are not recommended for weight reduction in clinical practice.*

4. Management of the metabolic syndrome through life habit changes

a. Weight control

ATP II (National Cholesterol Education Program 1993; 1994) recommended increased emphasis on weight reduction as part of LDL-lowering therapy for overweight/obese persons who enter clinical guidelines for cholesterol management. ATP III confirms this recommendation. However, in ATP III, emphasis on weight reduction is delayed until after other dietary measures are introduced for LDL lowering (reduced intakes of saturated fatty acids and cholesterol and possibly other options for LDL lowering [plant stanols/sterols and increased dietary fiber]) (see Figure V.2–1). The delay in emphasizing weight reduction is to avoid overloading new patients with a multitude of dietary messages and to concentrate first on LDL reduction. After an adequate trial of LDL-lowering measures, attention turns to other lipid risk factors and the metabolic syndrome (see Figure V.2–1). Weight reduction then becomes a major focus of TLC. In 1998, the NHLBI published Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults from the OEI (National Institutes of Health 1998a,b). This is an evidence-based report, and its recommendations for techniques of weight reduction are accepted by ATP III for persons undergoing management for cholesterol disorders. The ATP III report does not independently develop evidence statements beyond those in the OEI report. ATP III endorses the importance of weight control described in the OEI report. Indeed, weight control alone, in addition to lowering LDL cholesterol, favorably influences all of the risk factors of the metabolic syndrome.

b. Increased regular physical activity

ATP II also recommended increased emphasis on regular physical activity. In ATP III, the emphasis is reinforced with particular attention to its benefits for management of the metabolic syndrome. The recommendation for increased physical activity is introduced when TLC is initiated and the recommendation is reinforced when emphasis shifts to management of the metabolic syndrome (see Figure V.2–1). Physical inactivity is a major risk factor for CHD (U.S. Department of Health and Human Services. Physical activity and health . . . 1996b; Fletcher et al., 1996). It raises risk for CHD in several ways, notably by augmenting the lipid and nonlipid risk factors of the metabolic syndrome. It further enhances risk by impairing cardiovascular fitness and coronary blood flow. Regular physical activity can help reverse these adverse effects. It can have favorable effects on the metabolic syndrome and can reduce VLDL levels, raise HDL cholesterol and, in some persons, lower LDL levels. Regular physical activity lowers blood pressure and reduces insulin resistance. It also has been reported to reduce risk for CHD independently of standard risk factors. The evidence base for the recommendation of increased physical activity as part of cholesterol management is presented in the U.S. Surgeon General's Report on Physical Activity (U.S. Department of Health and Human Services. Physical activity and health . . . 1996b) and will not be detailed in this report. The purposes of regular exercise are to promote energy balance to maintain healthy body weight, to alleviate the metabolic syndrome, and to independently reduce baseline risk for CHD. In certain circumstances, a physician has the option of referring a patient to an exercise specialist for prescription and guidance in exercise training. Exercise specialists can complement nutrition professionals in implementation of the TLC diet by guiding individuals in a healthy exercise program.

5. Practical approach to life habit changes

a. Role of the physician

The physician is crucial to initiating and maintaining the patient's dietary adherence. Physician knowledge, attitude, and motivational skills will strongly influence the success of dietary therapy. A positive attitude combined with effective dietary assessment, initiation of therapy, and followup are essential for initial and long-term adherence. The physician should try to determine the patient's attitude towards acceptance of and commitment to TLC. The physician's key responsibilities include: assessment of CHD risk, dietary assessment, explanation of the problem for the patient, decision about appropriate therapeutic plan, and description of the plan to the patient. The multiple benefits of lifestyle changes should be emphasized. The need for lifestyle change, even when drugs are prescribed, should be stressed. In this section, one model for the role of the physician in the institution and followup of dietary therapy will be described. This model can be modified according to the constraints of the practice setting. The key feature of this model is the introduction of dietary therapy in a stepwise manner, beginning with an emphasis on lowering LDL cholesterol and followed by a shift in emphasis to management of the metabolic syndrome, if the latter is present. The essential steps in this model are shown above in Figure V.2–1.

1) Visit 1: Risk assessment, diet assessment, and initiation of therapeutic lifestyle change

Some persons do not qualify for immediate clinical management to lower LDL because their LDL level is not above the goal for their category of risk for CHD (see Section III). Nonetheless, the physican should appropriately control other risk factors, provide a public health message on overall risk reduction, and prescribe subsequent lipoprotein reevaluation as needed. Suggestions to assist the physician in conveying the public health message are outlined in Table V.1–3.

For persons who require dietary therapy, the first step is assessment of lifestyle habits. CAGE questions provide the physician with a way to rapidly assess current intakes of LDL-raising nutrients (Table V.2–4). A more detailed tool for both assessment and as a guide to TLC is available in Table V.2–6. Therapeutic change in the first visit should begin with the TLC diet. If the patient demonstrates a lack of basic knowledge of the principles of the TLC diet, the physician should consider referral to a nutrition professional for medical nutrition therapy.

2) Visit 2: Intensifying the TLC diet for LDL cholesterol lowering

Approximately 6 weeks after starting the TLC diet, lipoprotein analysis is repeated and assessed. If the LDL cholesterol goal is achieved by 6 weeks, the patient should be commended for his/her adherence and encouraged to continue lifestyle changes (Figure V.2–1). If the LDL goal has not been achieved, the LDL-lowering TLC should be intensified. Depending upon the patient's level of dietary adherence, various options exist. More vigorous reduction in saturated fats and cholesterol, adding plant stanols/sterols (2 g/day), increasing viscous fiber (see Table V.2–5), and referral to a nutrition professional can all enhance LDL lowering.

The physician should not ignore the power of TLC to reduce CHD risk. Despite the marked advances in drug therapy for elevated LDL cholesterol level, ATP III places increased emphasis on nutrition and physical activity for cholesterol management and overall risk reduction. The low prevalence of CHD in populations that consume low intakes of saturated fats and cholesterol and high intakes of other healthful nutrients, and who maintain desirable body weight through balanced caloric intake and output, illustrate what can be achieved without drug therapy (Keys 1984). Moreover, specifically for LDL cholesterol reduction, the combination of several dietary modifications can produce a reduction in LDL levels that rivals reductions produced by standard doses of statins. LDL cholesterol responses shown in Table V.5–2 represent conservative estimates based on the literature. Although cumulative responses have not been documented by clinical trial, a sizable summed response from the multiple components of TLC is likely.

Table V.5–2. Approximate and Cumulative LDL Cholesterol Reduction Achievable By Dietary Modification

Dietary Component	Dietary Change	Approximate LDL Reduction
Major		
Saturated fat	<7% of calories	8–10%
Dietary cholesterol	<200 mg/day	3–5%
Weight reduction	Lose 10 lbs	5–8%
Other LDL-lowering options		
Viscous fiber	5–10 grams/day	3–5%
Plant sterol/stanol esters	2g/day	6–15%
Cumulative estimate		20–30%

Adapted From Jenkins et al., 2000a.

3) Visit 3: Decision about drug therapy; initiating management of the metabolic syndrome

If the LDL cholesterol goal has not been achieved after 3 months of TLC, a decision must be made whether to consider adding drug therapy. If drugs are started, TLC should be continued indefinitely in parallel with drug treatment. Although the apparent ease of drug use is appealing, the additive effect of TLC to drug therapy in LDL cholesterol lowering is substantial and should not be overlooked. For example, Hunninghake et al. (1993) reported an extra 5 percent lowering of LDL cholesterol when lovastatin therapy was combined with dietary therapy. This additional LDL cholesterol lowering equates to doubling the dose of the statin, due to the log-dose characteristics of statin usage. Other studies revealed a much greater LDL reduction when dietary therapy plus plant stanols were combined with statin therapy (Gylling et al., 1997; Blair et al., 2000). These dietary options, if successfully implemented, are preferable to progressively increasing doses of LDL-lowering drugs.

A second purpose of Visit 3 is to initiate lifestyle therapies for the metabolic syndrome, if it is present. Emphasis in TLC shifts to weight control and increased physical activity. The principles of weight control are described in the Obesity Education Initiative report (National Institutes of Health 1998a,b).

Because of the complexities and frequent failures of long-term weight control in clinical practice, consideration should be given to referring overweight or obese individuals to a qualified nutrition professional for medical nutrition therapy.

A second element of treatment of the metabolic syndrome is to increase physical activity. The physician should provide specific recommendations for physical activity depending on the patient's physical well-being and social circumstances. Consideration also can be given to referral to an exercise specialist for guidance if this resource is available. Moderate, sustained exercise can cause a significant reduction in baseline risk for CHD. Examples of moderate intensity exercise that may be useful to individuals are listed in Tables V.2–6 and V.5–3.

Moderate intensity physical activity should be promoted for most people. Moderate amounts of vigorous activity also can be beneficial for some individuals, provided safety is ensured. Suggestions to incorporate more exercise into daily life are shown in Table V.5–4.

Table V.5–3. Examples of Moderate* Physical Activity in Healthy Adults†

- Brisk walking (3–4 mph) for 30–40 minutes
- Swimming—laps for 20 minutes
- Bicycling for pleasure or transportation, 5 miles in 30 minutes
- Volleyball (noncompetitive) for 45 minutes
- Raking leaves for 30 minutes
- Moderate lawn mowing (push a powered mower) for 30 minutes
- Home care—heavy cleaning
- Basketball for 15–20 minutes
- Golf—pulling a cart or carrying clubs
- Social dancing for 30 minutes

* Moderate intensity defined as 4–7 kcal/minute or 3–6 METS. METS (work metabolic rate/resting metabolic rate) are multiples of the resting rates of oxygen consumption during physical activity. One MET represents the approximate rate of oxygen consumption of a seated adult at rest, or about 3.5mL per min per kg.

† This table was adapted from the recommendations of the Surgeon General's Report on Physical Activity and Health (U.S. Department of Health and Human Services. Physical activity and health . . . 1996b) and the Centers for Disease Control and Prevention and American College of Sports Medicine (Pate et al., 1995).

Table V.5–4. Suggestions to Incorporate More Physical Activity into the Day

- Walk more—look for opportunities!
  - Park farther away in parking lots near a mall so you have a longer walk
  - Walk or bike if your destination is just a short distance away
  - Walk up or down 1–2 flights of stairs instead of always taking the elevator
  - Walk after work for 30 minutes before getting in the car and sitting in traffic
  - Walk home from the train or bus—take a longer route so it takes 20 minutes instead of 5–10
  - Walk with a colleague or friend at the start of your lunch hour for 20 minutes
- Do heavy house cleaning, push a stroller, or take walks with your children
- Exercise at home while watching television
- Go dancing or join an exercise program that meets several times per week
- If wheelchair bound, wheel yourself for part of every day in a wheelchair

4) Visit N: Long-term follow-up and monitoring adherence to therapeutic lifestyle changes (TLC)

The patient who has achieved the goal LDL cholesterol as a result of TLC must be monitored for the long term. TLC is maintained indefinitely and reinforced by the physician and, as

appropriate, by a nutrition professional if medical nutrition therapy is necessary. The patient can be counseled quarterly for the first year of long-term monitoring and twice yearly thereafter.

LDL cholesterol is measured prior to each visit, and the results are explained at the counseling session. When no lipoprotein abnormalities other than elevated LDL cholesterol are present, monitoring at 6-month intervals is appropriate. If elevated cholesterol level redevelops, the procedure outlined above for diet therapy of elevated LDL cholesterol should be reinstituted.

Persons who fail to achieve their goal LDL cholesterol by dietary therapy can be classified as having an inadequate response to diet. Such responses fall into four categories:

- *Poor adherence*. Some persons adhere poorly to diet modification despite intensive and prolonged dietary counseling. They are not ready to change for various reasons. Physician endorsement of the importance of diet is essential for facilitating increased interest on the part of the patient. If the patient admits a lack of willingness to change diet or other life habits, drug therapy may be the only reasonable option to effectively lower LDL.
- *Gradual change*. Some individuals modify eating habits only gradually. They may adhere poorly to diet in the first few months but eventually will modify their eating habits to meet the goals of therapy. Up to a year of instruction and counseling may be required for these persons. This is especially true for persons who are following a weight reduction plan. Ongoing follow-up and reinforcement is crucial for developing long-term adherence. A continued effort to achieve adherence to life-habit changes should not be abandoned if drug therapy is started.
- *Poor responders*. A minority of persons are non-responders to dietary therapy and will have high LDL cholesterol levels that are inherently resistant to dietary modification despite good adherence (Denke 1994, 1995; Denke and Grundy, 1994). The mechanisms for this resistance are not well understood. Recognition of such persons is important, and care must be taken not to accuse them of failing to adhere to diet when they are non-responders. Drug therapy may be the only effective means of treatment of high blood cholesterol in such persons, but continued adherence to TLC is helpful for maintaining an overall healthful dietary pattern.
- *Inadequate responders*. Persons with severe elevations of LDL cholesterol often do respond to dietary therapy, but the cholesterol lowering achieved is inadequate to reach the LDL cholesterol goal. For such persons, a 3-month period of intensive diet therapy before adding drugs is not necessary.

b. Role of nurses, physician assistants, and pharmacists

Other health professionals associated with the physician facilitate patient management. The role of nutrition professionals is addressed in more detail below. Other health professionals—nurses, physician assistants, nurse clinicians, pharmacists, and other professionals—can participate in patient education (e.g., explaining the rationale for dietary change, goal setting, selection of appropriate foods, diet adherence), promoting behavioral changes, and monitoring dietary changes. These health professionals should receive appropriate training in dietary assessment, dietary education, and counseling. Hospital nurses play a vital role in guiding patients during

hospital admissions for acute coronary events. NCEP and AHA offer various educational materials to assist in training health professionals.

c. Specific role of registered dietitians and other qualified nutrition professionals

Registered and/or licensed dietitians are certified providers of medical nutrition therapy (MNT), and qualify for Medicare reimbursement. Individual state licensure laws have established credentials for determining qualifications for nutrition counselors. Dietitians with expertise and experience in dietary counseling for lipid lowering can be especially effective in facilitating adherence to TLC. Registered dietitians and other licensed nutritionists can be located through local hospitals and state and district affiliates of the American Dietetic Association. The American Dietetic Association (www.eatright.org; 216 W. Jackson Blvd., Suite 800, Chicago, IL 60606-6995; 312-899-0040) maintains a roster of dietitians and responds to requests in writing or e-mail for assistance in locating a registered dietitian in a given area. Dietitians with particular expertise in cholesterol management are available in most large medical centers where they are often part of a multidisciplinary lipid clinic or cardiac rehabilitation team.

Medical nutrition therapy provided by a registered dietitian is a service that involves a comprehensive assessment of a patient's overall nutritional status, medical data, and diet history, followed by intervention to prescribe a personalized course of treatment.

The following Medical Nutrition Therapy CPT Codes can be found in the American Medical Association Current Procedural Terminology CPT 2000, pg. 300:

- 97802 Medical nutrition therapy; initial assessment and intervention, individual face-to-face with the patient, 15 minutes each
- 97803 reassessment and intervention, individual face-to-face with the patient, 15 minutes each
- 97804 group (2 or more individual(s), 30 minutes each

(For medical nutrition therapy assessment and/or intervention performed by a physician, see Evaluation and Management or Preventive Medicine service codes.)

CPT codes currently cover consideration of MNT for management of diabetes mellitus and renal disease.

1) Role of the nutrition professional in LDL-lowering therapy

When the physician chooses to consult a nutrition professional (dietitian) at Visits 1 or 2 for medical nutrition therapy, the goal is to enhance adherence to TLC. Medical nutrition therapy should start with dietary assessment, including the patient's motivational level and willingness to change. A dietary assessment questionnaire, MEDFICTS, which was originally developed for and printed in ATP II (National Cholesterol Education Program 1993,1994) is included in Diet Appendix A. Other cardiovascular dietary assessment tools are also available (Ammerman et. al., 1991; Connor et al., 1992; Gans et al., 1993; Kris-Etherton et al., 2001; Kristal et al., 1990; Retzlaff et al., 1997; Peters et al., 1994). Proper assessment leads to a tailored dietary

prescription. This prescription then goes to the physician, who can encourage adherence and monitor progress.

a) First: dietary assessment

A thorough and detailed assessment of the patient's knowledge, attitudes, and behavior regarding diet is essential for effective nutrition counseling. Assessment requires attention to dietary history, cultural influences, and current eating habits. It also includes recording the patient's weight and weight history, BMI, and waist circumference. The presence of abdominal obesity points to the metabolic syndrome. To assess current eating habits, the following information is needed:

- What times of the day does the patient usually eat?
- Are some meals routinely skipped?
- At what time does the patient eat his/her largest meal?
- Where are meals typically prepared and eaten (e.g., in a restaurant, work cafeteria, fast-food restaurant, deli, at home, or in the homes of others)?
- Are there occasions when stress increases food consumption?
- Are meals eaten at home purchased out and brought in, prepared from processed pre-packaged foods, or prepared fresh from the market?
- Which are favorite foods and what foods are disliked?
- Who is responsible for food shopping and preparation?
- What foods will be most difficult to increase or decrease?
- How well does the patient recognize serving sizes?

The nutrition professional should assess the patient's general knowledge of nutrition as it relates to elevated LDL cholesterol, the ability to read labels, educational level, motivation, attitudes toward diet, and the extent to which family members can facilitate dietary changes.

b) Dietary guidance on adopting the TLC *Diet*

To help patients adapt to the TLC Diet, the dietitian can:

- Focus on dietary patterns to facilitate LDL lowering. These patterns are consistent with the Dietary Guidelines for Americans 2000 (U.S. Department of Agriculture . . . 2000) to achieve overall health and to further reduce baseline risk for CHD. This eating pattern is recommended for the entire family.
- Seek mutual agreement on an overall plan for diet modification as well as specific foods and eating habits that need to be changed. Emphasis goes first to dietary habits that affect LDL cholesterol levels. Highest on the list are foods rich in saturated fatty acids and cholesterol. The dietitian can review options for choosing preferred foods that lower LDL levels. The need for self-monitoring is reinforced; and simple approaches to tracking saturated fat, fiber, fruit, and vegetable intake are provided. Weight reduction includes learning how to control portion sizes. Also, documenting preparation and the quantities of

different foods helps in long-term adherence. Practical teaching with measuring cups, spoons, food models, or even a food scale will enhance patient understanding. Keeping a food record during weekends and weekdays can facilitate discussion with the dietitian. Electronic (e-mail) links between dietitian and patient may enhance checking food records or reporting self-monitoring activities.

- Help patients identify sources of saturated fat in their usual diet, especially "hidden" fats in foods, such as baked goods, cheese, salad dressings, and other processed foods. Advice on alternative food choices, including snack foods, should be provided. For persons willing to prepare foods at home, appropriate techniques and cooking methods can be addressed. For those who eat out regularly, guidance on how to select from a menu and purchase premade take-out food should also be given.
- Apply motivational interviewing techniques to provide encouragement and to empower patients to choose wisely on different eating occasions. Gradual, step-wise changes in current eating habits are more likely to achieve long-term adherence than drastic changes. Starting with a specific food or food group, such as the type of milk used, how to reduce portion size of meats, how to substitute egg whites for whole eggs in baking, or how to use margarines and oils in the place of fats rich in saturated fatty acids are excellent topics to pursue. The dietitian should involve other individuals of significance (e.g., parents, spouse, and children) in dietary instructions.
- Recommend a variety of foods from all food groups to help achieve adequate nutrient intake: vegetables, fruits, grain products, potatoes and legumes, dairy products, and lean meat, poultry, and fish. Use of specially prepared processed foods, fat-free or fat modified snacks, desserts, etc. is not necessary, although some persons find these food choices appealing.
- Promote use of the Nutrition Facts food label to help patients learn to gauge saturated fat and cholesterol intakes. Saturated fat amounts listed on the Nutrition Facts food label correspond to 10 percent of calories; still lower intakes are needed to attain <7 percent. Persons should should be taught to routinely read the labels of all processed foods.

c) Specific foods and preparation techniques

Recommended food choices for the TLC Diet are summarized in Table V.2–6. This diet can be both tasty and nutritious. Many choices of high-quality and recommended foods are available in supermarkets, restaurants and as take-out options.

To decrease intake of saturated fat, total fat, and cholesterol, the emphasis of the diet should be on consumption of vegetables; fruits; breads, cereals, rice, legumes, and pasta; skim milk and skim milk products; and poultry, fish, and lean meat. There are many different eating styles in the United States that reflect diverse cultures and practices. Special attention to unique dietary preferences based on diverse cultures and eating habits can facilitate adoption of the TLC diet. Sample menus are presented in Diet Appendix B.

Food preparation techniques should emphasize lower fat cooking and preparation methods (broiling, baking, grilling, steaming, poaching without added fat, trimming fat from meat, draining fat after cooking, and removing skin from poultry). Liquid vegetable oils high in unsaturated fatty acids (e.g. canola, corn, olive, rice bran, safflower, soybean, sunflower) are

recommended in moderation. Since the major sources of saturated fat and total fat in the American diet are meat and high-fat dairy products, and since these foods as well as eggs are the major sources of dietary cholesterol, persons should limit consumption of foods containing butterfat such as whole milk (3.5 percent fat) and even reduced fat (2 percent) milk, butter, cheese, ice cream, cream, and pizza; fatty meats such as regular ground beef (hamburger), processed meats (hot dogs, sausage, bacon), and high-fat luncheon meats (bologna, salami, chopped ham products), as well as poultry skin. Low-saturated-fat substitutes, such as fat-free or 1 percent milk, soft margarine, low-fat cottage cheese, or low-fat or fat-free "ice cream" can be used. Egg yolks should be limited to 2 per week. Organ meats (liver, brain, sweetbreads) are rich sources of cholesterol and should be limited. Of the shellfish, only shrimp is moderately high in cholesterol and inclusion in the diet should be guided by the daily dietary cholesterol allowance. The vegetable oils rich in saturated fat—coconut oil, palm kernel oil, and palm oil—are used in some commercial foods and food products. Choose products that are labeled low saturated fat, e.g., 1 gram of saturated fat per serving, and meats that are labeled as lean.

Although persons need not purchase special foods for implementation of the TLC Diet, many new fat-modified products on the market may facilitate adherence to the TLC Diet.

d) Recommendations by food group

The following information about specific food choices can help persons adopt the TLC Diet.

- Breads, cereals, pasta, whole grains, potatoes, rice, dry peas, and beans (6 or more servings per day). These foods are high in complex carbohydrates and fiber, provide protein, and also are generally low in saturated fat, cholesterol, and total fat. Dry beans and peas are good sources of plant protein and are fiber-rich. They should be substituted for foods high in saturated fat, cholesterol, and total fat. Cereals can be eaten as snacks as well as for breakfast. Dry peas, beans, and legumes can be used in nutritious, tasty, lower fat entrees or accompaniments. Pasta, potatoes, rice, and vegetables can be combined with smaller amounts of lean meat, fish, or poultry for a tasty main dish that can provide less saturated fat and calories.
- Fruits and vegetables (5 or more servings per day). Fruits, vegetables, or both should be emphasized at each meal. They are major sources of vitamins C, E, and A, beta-carotene, other vitamins, fiber, and some minerals, and contribute to achieving the recommended allowances of these nutrients. Snacks and desserts that feature fruits and/or vegetables can be low in saturated fat, total fat, and cholesterol, and very nutritious.
- Fat-free or 1 percent dairy products (2–3 servings per day). Dairy products are important sources of protein, calcium, phosphorus, and vitamin D. Fat-free milk and other fat-free or low-fat dairy products provide as much or more calcium and protein than whole milk dairy products, with little or no saturated fat. Fat-free milk or 1 percent fat milk, fat-free or low-fat cheese (e.g., 3 g per serving), 1 percent fat cottage cheese or imitation cheeses made from vegetable oils, and fat-free or low-fat yogurt are good choices. It should be noted that 2 percent fat dairy products are still rich in saturated fat. Evaporated fat-free milk can be used in recipes calling for heavy cream. Low-fat or fat-free yogurt, 1 percent fat cottage cheese, and fat-free sour cream substitutes can replace sour cream in dips and salad dressings.

- Lean meats (beef, pork, and lamb), poultry, and fish (up to 5 oz per day). Lean cuts of beef include sirloin tip, round steak, rump roast, arm roast and, for pork, center-cut ham, loin chops, and tenderloin. All visible fat should be trimmed before cooking. Ground meat should be extra-lean and drained well after cooking. Meat can be ground at home or a butcher can grind very lean, well trimmed cuts of meat such as those that come from the round. Ground turkey, which can be seasoned and used like ground beef, is very lean if it does not contain turkey skin and fat. Both lean ground meat and ground turkey can be incorporated into soups, stews, and casseroles that contain grain products and vegetables. Special reduced-fat ground meat products (e.g., with carrageenan) may be selected. It is not necessary to eliminate or drastically reduce lean red meat consumption. Lean meat is rich in protein, contains a highly absorbable iron (Fe^{++}), and is a good source of zinc and vitamin B12. Lean meat can contribute to maintenance of iron stores in premenopausal women.
  - Foods containing soy-based meat analogues can be substituted in part for meat products.
  - Processed meats. Processed meats, such as lunch meat, bacon, bologna, salami, sausage, and frankfurters generally have a high content of saturated fat and sodium. Several new processed meat products are lower in saturated fat, total fat, and cholesterol. Read the Nutrition Facts food label to choose foods low in saturated fat, cholesterol, and sodium.
  - Organ meats. Liver, sweetbreads, kidneys, and brain have a high cholesterol content and should be used only occasionally.
  - Chicken and turkey. These are good sources of lean protein. Removing the skin and underlying fat layers substantially reduces the fat content. Chicken and turkey can be substituted for some of the lean red meat in the diet, but they do not contain as much iron. Chicken and other poultry should be prepared in ways that minimize the addition of saturated fat.
  - Fish. Fish are low in saturated fat, some are high in n-3 fatty acids (see Diet Appendix C), and they are a good source of lean protein. The preparation of fish is important. Like chicken and turkey, it should be prepared to limit additional saturated fat.
  - Shellfish. Shellfish are low in saturated fat. The cholesterol content of shellfish is variable (see Diet Appendix C). Shrimp are relatively high in cholesterol, but can be eaten occasionally. About 5 ounces of fish, poultry, or meat per day can be included on the TLC Diet as 2 servings, each serving about the size of a deck of playing cards. A serving of meat in a restaurant often exceeds 5 ounces. (The saturated fat, total fat, and cholesterol content of various cooked meats are presented in Diet Appendix C).
- Fats and oils (including fats and oils used in food preparation). Fats high in saturated fat, *trans* fat, and cholesterol must be limited. This includes lard and meat fat. Some vegetable fats—coconut oil, palm kernel oil, and palm oil—are high in saturated fat and should be avoided; they often are used in bakery goods, processed foods, popcorn oils, and nondairy creamers. The Nutrition Facts food label is a guide for choosing fats and oils lowest in saturated fat. Hydrogenated shortenings and hard margarines are sources of *trans* fat and should be reduced. Vegetable oils and fats high in unsaturated fat do not raise blood cholesterol, but they have a high caloric density. These include canola oil, corn oil, olive oil, safflower oil, soybean oil, and sunflower oil. Margarine contains some *trans* fat but has less cholesterol-raising potential than butter, and thus is preferable to

butter. In general, the softer the margarine, the less LDL-cholesterol-raising potential it has. Hydrogenated shortening contains *trans* fat, resembles hard margarines, and should be limited. Hydrogenated shortenings are found in many commercially prepared baked foods, such as crackers, cookies, doughnuts, and desserts. There are many reduced fat margarines, vegetable oil spreads, and low-fat and fat-free salad dressings on the market. The Nutrition Facts food label provides the amount of fat and saturated fat per serving.

- Nuts. Nuts are high in fat, but in most nuts the predominant fats are unsaturated. The intake of nuts should fit within the calorie and fat goal.
- Eggs. Egg yolks are high in cholesterol (~215 mg/egg) and should be limited to no more than two egg yolks per week. Egg yolks often are found in cooked and processed foods. Egg whites contain no cholesterol, and they can be eaten often. Egg whites or commercial egg substitutes or reduced-cholesterol egg products can replace whole eggs in many recipes.

e) Other eating tips

- Snacks. Some choices for snacks that are low in saturated fat are graham crackers, rye crisp, melba toast, pretzels, low-fat or fat-free crackers, bread sticks, bagels, English muffins, fruit, ready-to-eat cereals, and vegetables; fat-free corn chips and potato chips can be made at home or purchased in some stores. Popcorn should be air popped or cooked in small amounts of vegetable oil. Low-fat cookies include animal crackers, fig and other fruit bars, ginger snaps, and molasses cookies.
- Desserts and sweets. Moderate amounts of sweets and modified-fat desserts (low in saturated fat) may be chosen. For example, fruits, low-fat or fat-free fruit yogurt, fruit ices, sherbet, angel food cake, jello, frozen low-fat or fat-free yogurt, and low-fat ice cream. Cookies, cakes, and pie crusts can be made using unsaturated oil or soft margarines, egg whites or egg substitutes, and fat-free milk. Candies with little or no fat include hard candy, gumdrops, jelly beans, and candy corn. Read the Nutrition Facts food label to choose those products lowest in saturated fat and calories.
- Cooking methods. Methods that use little or no fat include steaming, baking, broiling, grilling, or stir frying in small amounts of fat. Cook foods in the microwave or in a nonstick pan without added fat. Foods may be pan fried with limited fat. Soups and stews should be chilled for a few hours, and the congealed fat removed. Salt should be limited in the preparation of soups, stews, and other dishes. Herbs and spices can often be used instead of salt to help prevent or control high blood pressure.
- Eating away from home. Choose entrees, potatoes, and vegetables prepared without sauces, cheese, or butter when eating away from home. Eat only a small portion of meat. Choose vegetable or fruit salads, with salad dressings on the side. Limit toppings, such as chopped eggs, crumbled bacon, and cheese. Request soft margarine instead of butter, and use it sparingly.

A reference work on food and nutrition may be useful to patients. One available reference is the USDA's Home and Garden Bulletin No. 72, Nutritive Value of Foods 399. In addition, a typical 1-day menu for TLC Diets for both men and women which displays different eating patterns is included in Diet Appendix B.

2) Role of the dietitian in management of the metabolic syndrome

After LDL cholesterol is controlled, medical nutrition therapy turns attention to the metabolic syndrome. Strategies for weight reduction described in the Obesity Education Initiative report (also see www.nhlbi.nih.gov) are helpful. Weight reduction and dietary change introduced in medical nutrition therapy aim to achieve and maintain goals for LDL cholesterol as well as glucose and blood pressure. Hypocaloric diets, increased physical activity, and weight loss usually improve levels of LDL cholesterol, glycemic levels, and blood pressure and have the potential to improve long-term metabolic control. The distribution of calories from total fat and carbohydrate can vary (see Table V.2–2) and can be individualized based on the nutrition assessment and treatment goals.

6. Improving patient adherence to life habit changes

Outpatient studies show that variability in lipoprotein responsiveness to diet is often due to poor compliance. Good compliance is hampered in part by increased consumption of foods prepared away from home. In 1995 about 40 percent of the food budget was spent on food prepared away from home, compared with 25 percent in 1970 (Lin et al., 1999). The consumer has less knowledge of and less control over the nutritional content of food prepared away from home. Moreover, calories, saturated fat, and cholesterol tend to be higher in premade food than food prepared at home (Lin et al., 1999). Food prepared away from home usually does not carry nutrition labeling. Barriers to adherence to dietary therapy must be addressed and reasonable solutions provided. Physicans in general report little confidence in the patients' ability to adhere to dietary change. In one survey, 17 percent of physicians reported that most patients complied, 59 percent reported that some complied, and 22 percent estimated that few patients complied.

Lack of adequate nutrition education in medical schools has been a contributing factor to low adherence to dietary therapy that fortunately is now being addressed. The newly implemented NHLBI-funded Nutrition Academic Award is now underway in 21 U.S. medical schools. This program provides training in nutritional assessment and counseling for medical students and other health professionals in training (Pearson et al., 2001). Other barriers, such as lack of time, lack of adequate referral strategies, lack of third party reimbursement, and competition with pharmacological intervention are also being addressed (Van Horn and Kavey 1997).

Beyond these systemic problems, a validated methodology related to effective nutritional assessment and intervention is lacking. Ready access to a brief dietary assessment tool and accompanying follow up assessments are as yet not standard practice for most physicians. Advances have been made in the past decade regarding the combined use of behavioral strategies along with standardized diet assessment and intervention approaches (Ammerman et. al., 1991; Connor et al., 1992; Gans et al., 1993; Kris-Etherton et al., 2001; Kristal et al., 1990; Retzlaff et al., 1997; Peters et al., 1994)(See Appendix A for an example of a validated assessment tool.)

There is growing evidence from the behavioral therapy literature that strategic approaches to lifestyle intervention can achieve better and more consistent long-term adherence (Glanz et al., 1994, Shepherd et al., 1987, Prochaska et al., 1994). These strategies are based on learning principles that address the need to overcome barriers to adherence with lifestyle change and reinforce newly adopted behaviors (Bandura 1997; Baranowski 1997; Glanz et al., 1994). The

vast majority of these studies appear in the weight management field (Perri 1998). The Obesity Guidelines panel reviewed 36 randomized clinical trial reports to determine potential benefits of behavioral therapy (National Institutes of Health 1998a,b). Key findings from these studies are summarized below:

- Multimodal strategies work better than a single approach
- More frequent contact is associated with better adherence
- Adherence declines with discontinued intervention or follow-up
- Greater intensity of intervention, especially initially, is associated with faster and more sustained adherence
- Motivation is enhanced when the patient sets achievable goals.

Further lessons learned from the behavioral literature emphasize the importance of baseline assessment of dietary intake, use of self-monitoring to improve adherence, and use of health messages that are matched to level of readiness to change, culturally sensitive, interactive, address prior knowledge, come from reliable sources, and recommend reasonable, gradual, and easily implemented change. Additional research is needed with measures of the efficacy and effectiveness of office-based dietary assessment methodology, especially as this relates to behavioral strategies enhancing dietary adherence.